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in geographic areas where this resistance appears to be endemic in other species such as *Klebsiella pneumoniae*.

Other resistance traits have been described in *E. coli* clinical isolates as plasmid-mediated quinolone resistance due to *qnr* and *aac(6')-Ib-cr* genes, and the efflux pump QepA. Moreover, production of plasmid 16S rRNA methylases has recently drawn attention as a novel aminoglycoside resistance mechanism in pathogenic gram-negative bacteria including *E. coli*. It confers high-level resistance to all aminoglycosides that are currently available.

Multiresistance in *E. coli* affects almost all antimicrobial families, it is easily transmitted through successful and virulent clones and can be spread from and among not only humans but animals and food. The role of continuous antimicrobial pressure in this phenomenon is unquestionable and requires control measures to curtail the spread and maintenance of these multiresistant isolates with high likelihood of causing serious and almost untreatable infections.

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20.002

Multidrug resistance in *Klebsiella pneumoniae*

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Hospital-acquired and clinically-important Gram-negative pathogens remain mostly *Enterobacteriaceae*, *Pseudomonas aeruginosa* and *Acinetobacter baumannii*. Among those Gram negatives, *Klebsiella pneumoniae* remains an important source of hospital spread of multidrug resistance. Wide-spectrum !-lactamases are increasingly reported in *Enterobacteriaceae* being either clavulanic-acid inhibited extended-spectrum !-lactamases (ESBLs) or carbapenem-hydrolyzing !-lactamases (CHBLs). Although first reported in *Klebsiella pneumoniae* mostly from 1980's to 2000's, ESBLs are developing rapidly among communityacquired *Escherichia coli*. These novel ESBLs of the CTX-M-type are reported worldwide with important structural and genetic diversity. Those ESBL genes may be transmitted from *E. coli* to *K. pneumoniae* providing a novel source of hospital-acquired multidrug-resistant *K. pneumoniae* since there are associated to other plasmid-mediated resistance determinants. The CHBLs identified in *Enterobacteriaceae* are mostly metallo-!-lactamases (Ambler class B enzymes) of the VIM/IMP-types in hospital-acquired *K. pneumoniae*. The Ambler class A carbapenemases of the KPC-type are also identifed mostly in *K. pneumoniae*, first from the USA and then worldwide. The latest reported CHBL in *K. pneumoniae* is OXA-48 mostly from Mediterranean countries. All this carbapenemase producers are difficult to detect in a clinical laboratory and may the source of multidrug resistance leading to therapeutic deadend. *K. pneumoniae* will remain the most important enterobacterial species as a source of multidrug resistance in hospital-acquired Gram negative isolates.

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Pseudomonas aeruginosa

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Evolution of antimicrobial resistance in *Acinetobacter baumannii:* Factors affecting multiresistance

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Acinetobacter baumannii are an important cause of nosocomial infections mainly in patients in the intensive care units. In this presentation I will analyse the evolution of antimicrobial resistance, the molecular bases associated with the increase in antimicrobial resistance, the factors affecting multiresistance and the current treatment of *Acinetobacter* infections.

Antimicrobial resistance has steadily increased in the last decade. Nowadays *A. baumannii* clinical isolates resistant to all antimicrobial agents even to colistin (panresistant) have been isolated in the nosocomial setting. Three major factors favour the acquisition of multiresistance: 1. Intrinsic resistance, mainly related to the interplay between decreased permeability (small number of porins) and constitutive expression of efflux pump(s) (AdeIJK, CraA); 2. Persistence in the environment, in this sense, biofilm-producing *A. baumannii* clinical isolates survive in inanimate surfaces longer than those non-producing biofilm. 3. Acquisition of genetic elements. It has recently been shown that resistance islands with a variable composition of resistance determinants interspersed with transposons, integrons and other genetic elements play an important role in the acquisition of multiresistance. However, this is not an universal contributor to multiresistance since target mutations, overexpression of efflux pumps, and IS elements located upstream from some resistance genes have also been found to be implicated in multiresistance. Although some clinical isolates are still susceptible to carbapenems and colistin, and therefore these antimicrobial agents can continue to be used, few options are available to treat infections caused by this microorganism. Tygecycline has been used to treat infections caused by *A. baumannii*. However, emergence of resistance to this antimicrobial agent has been reported during treatment when this monotherapy.

This microorganism, albeit with slight differences depending on the country, presents resistance to multiple antimicrobial agents, occasionally including resistance to colistin, hence, it can be considered the paradigm of nosocomial multiresistant bacteria.

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